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CHOLESTEATOSIS OF THE ATTIC*

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I FEEL greatly honoured to have been asked to address you on the old subject of cholesteatomatous otitis media. I must confess that I am not sufficiently familiar with the term "cholesteatosis of the attic" to be able to give it a precise definition.

We differentiate between chronic otitis media with a central perforation and that with an upper marginal perforation where the tympanic defect leads into the attic. Cholesteatomas occur as a rule only in the latter type of middle-ear infection and the cholesteatomatous lamellae in the attic can be seen through the upper marginal perforation. Cholesteatomas in the middle ear may be classified into two groups according to the extent of the perforation. The larger of these includes cases with *large* upper marginal perforations, while the smaller group includes the less common cases with *small* upper anterior or posterior marginal perforations. In 1934 statistical analysis of 763 cases of cholesteatomatous otitis media seen at the Zürich ear clinic placed 631 cases into the first category with large upper marginal drum defects, while only 132 cases fell into the second category with small marginal perforations.

Intracranial complications occurred in only 1·7 per cent. of the larger group of cholesteatomas with *extensive* drum defects. While the classical radical operation obviates this danger, the unpleasant otorrhoea tends to continue in many patients, and hearing often deteriorates after the radical

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mastoidectomy. These unsatisfactory results have prompted many otologists to adopt conservative methods in the management of these cases. The large tympanic defects enables one to reach the cholesteatoma relatively easily and to remove the masses of dead epithelium from the middle ear. As a result of this the discharge from the ear usually stops, but, of course conservative treatment cannot hope to improve the hearing. At best the patient will remain under medical supervision for the rest of his life.

In the smaller group of cholesteatomas with *small* upper marginal perforations the incidence of intracranial complications is four times higher (6 per cent. as compared with 1·7 per cent.). Conservative treatment cannot reach the affected area through the small drum defect and the danger of intracranial complication continues. Radical mastoidectomy is, therefore, the treatment of choice for these cases and has been recognized as such since the days of H. Schwartze, L. Stacke, K. Wittmaack *et al.* The institution of early surgery is amply justified by results. A conservative radical operation not only eradicates that form of cholesteatoma and its inherent danger to life, it will also preserve or even improve hearing and, as a rule, halt the unpleasant discharge from the ear.

It is to be expected that the technical advances of temporal bone surgery, and especially fenestration surgery, should lead to better results in the treatment of all forms of cholesteatoma. However, an exact knowledge of the pathogenesis of cholesteatomas is essential for the successful application of these new surgical techniques. In the Zürich series of cholesteatomas with large drum erosions, the middle-ear suppuration started as a necrotizing otitis media of infancy in 35 per cent. of cases. This arose in the course of scarlet fever, measles, diphtheria, tuberculosis or influenza. In the remaining 65 per cent. of cholesteatoma with large perforations no such history could be obtained. But in the majority of cases the middle-ear infection dated from early childhood and it may be assumed that these cholesteatomas also began as a necrotizing otitis media of unknown ætiology in infancy. J. Habermann was the first to observe the reaction of the epithelium of the external auditory meatus abutting on the attic perforation during an attack of necrotizing otitis media in infancy. As a result of this reaction meatal epithelium grows inwards through the perforation and once in the middle ear stimulates the production of a cholesteatoma. Some supporters of this immigration theory regard this ingrowing of the meatal epithelium as an attempt to repair the destroyed mucosa of the middle ear. P. Manasse has pointed out, however, that the cholesteatomatous matrix shows a strong tendency to proliferation, which is unlike the simple process of epithelial covering of the cavity. The invading squamous epithelium is not content to advance on the surface. It spreads mainly by sending forth strands and processes into the submucous connective tissue of the still intact cubical epithelium of the middle ear. Thus from its very beginning the development of cholesteatoma is not to be

looked upon as a replacement of the destroyed mucosa of the middle ear by squamous epithelium. Rather is it to be seen as a struggle between the two types of epithelium. This struggle is rather an unequal one, Fig. 1, the cholesteatoma undermines the mucosal cubical epithelium, lifting it off the underlying connective tissue and pushing it away as it advances. The cholesteatomatous process frequently breaks through the intact mucosa

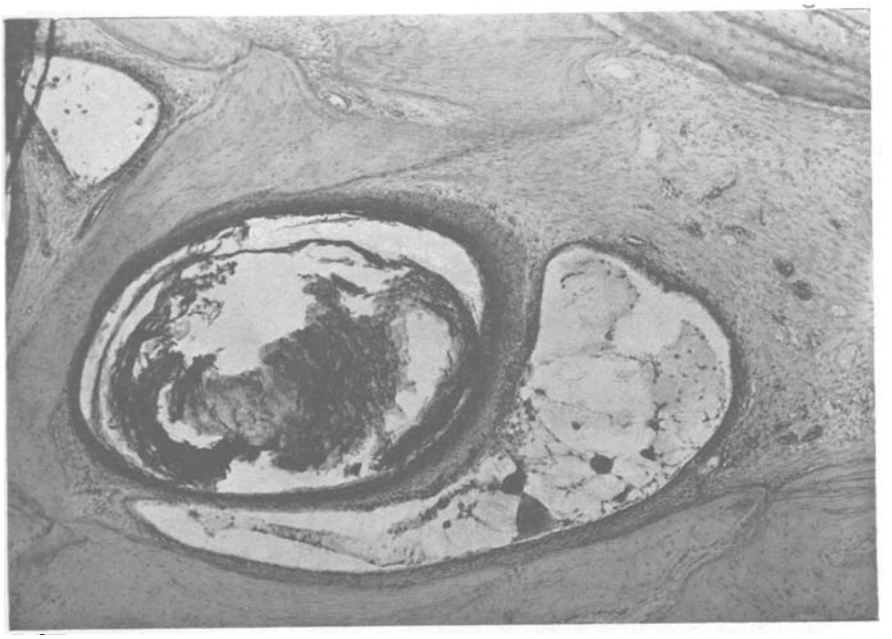

FIG. 1.

Otitis media chronica cholesteatomatosa in the case of an 11 year old boy. The cholesteatoma undermines the mucosal cubical epithelium.

of the middle ear and (Fig. 2) once on the surface masses of desquamated squamous epithelium are shed into the cellular system. The mucosa reacts to this by producing an inflammatory exudate. The single layered cubical epithelium becomes heaped up into cushions many layers thick. The aggressive behaviour of the cholesteatomatous matrix in relation to the mucosa in cases with a large drum defect cannot therefore, be regarded as a simple harmless attempt at healing. On the other hand the matrix does not attack the surrounding bone (Fig. 3). In the early stages of the disease the bony tissues respond to the inflammatory stimulus by laying down new bone and by ossifying the submucous connective tissue. The active growth of the matrix in the depths of the submucosa, proceeds in the same connective tissue plane alongside the osteogenetic process, without either process interfering with the other. When all the available submucous connective tissue has been used up, the cholesteatomatous

FIG. 2.
Otitis media chronica cholesteatomatosa in the case of an 11 year old boy. The cholesteatoma breaks through the mucosa of the inner ear.

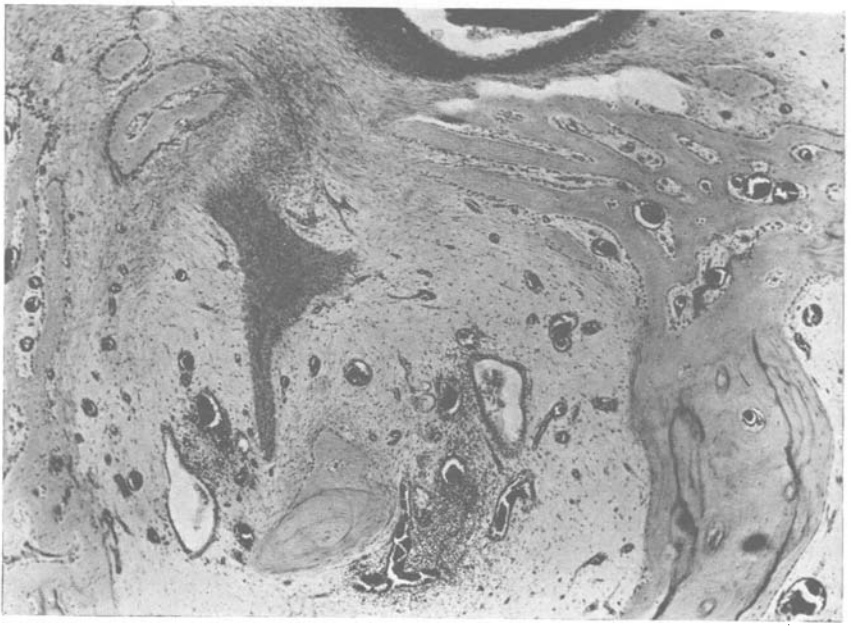

FIG. 3.
Otitis media chronica cholesteatomatosa in the case of a 23 year old man. The active growth of the matrix proceeds in the connective tissue alongside the osteogenetic process taking place in the same connective tissue.

channels become choked with desquamated squamous epithelium. It is only at this stage that the bed of the cholesteatoma begins to enlarge passively, the secondarily laid down sclerotic bone being reabsorbed as a result of direct pressure by the mass of the cholesteatoma. The passive expansion at the expense of surrounding bone, which is characteristic of the advanced cholesteatoma, is less important for the understanding of the whole disease than the early active growth of cholesteatomatous processes within the submucous connective tissue. There can be little doubt from the above observations that cholesteatomas with large tympanic perforations arise as a result of an inflammatory reaction causing immigration of the meatal epithelium.

On the other hand there is no unanimity of views concerning the pathogenesis of the rarer cholesteatomas with *small* upper marginal perforations. In the Zürich series there were 132 such cases and only about 20 per cent. gave a history of otitis media in early childhood. In none of the cases did an ear specialist observe the appearance of a small upper marginal perforation in the course of the otitis media. At the onset the disease is usually symptomless, without an obvious association of a middle ear infection. The small perforation is often discovered accidentally in the course of a routine examination. The character of the perforation at this early stage already shows signs of a cholesteatoma having become established to it. Operation on such early cases reveals a surprisingly large cholesteatoma in the attic. It is tempting to compare these cases with the very rare cholesteatomas with an intact ear drum, which are occasionally encountered during operations for other aural conditions.

Regarding this possibility M. Diamant states that "the invasion of the stratified squamous epithelium from the auditory meatus is impossible". He considers that these cholesteatomas arise, as suggested by R. W. Teed, from embryonic squamous epithelial remnants in the mucosa of the middle ear. Dan McKenzie speaks of "a primary tumour, similar to intracranial cholesteatoma", while as early as 1925 F. R. Nager pointed out that "the true cholesteatomas are so rare as to be negligible". The almost symptomless origin of some cholesteatomas is explained in the Anglo-Saxon literature by postulating a metaplasia of the mucosa of the middle ear to a keratinizing stratified squamous epithelium as a result of inflammatory changes. R. R. Simpson considers that this metaplasia may be induced by the formation of a cholesterol granuloma in the middle ear and his views are shared by A. Tumarkin.

Can the pathogenesis of these cholesteatomas of insidious onset and with small upper marginal perforations be explained on the basis of the immigration theory?

F. Bezold's hypothesis of a chronic tubal catarrh leading to retraction and finally rupture of Shrapnell's membrane finds little support today. While retraction of the pars flaccida is a common occurrence, its rupture

with consequent cholesteatoma formation has never been observed to the best of our knowledge. There is no reason to suppose that such a perforation would not heal rather than lead to a cholesteatoma. F. Bezold himself was aware of this difficulty and pointed to the co-existing inflammatory factors as maintaining the perforation in Shapnell's membrane. P. Manasse drew attention to the proliferative powers of the drum around small, upper marginal perforations in the presence of chronic or subacute infection. J. Habermann considered this a possible factor in the aetiology of the cholesteatomas. Finally, W. Lange furnished histological proof of deeply

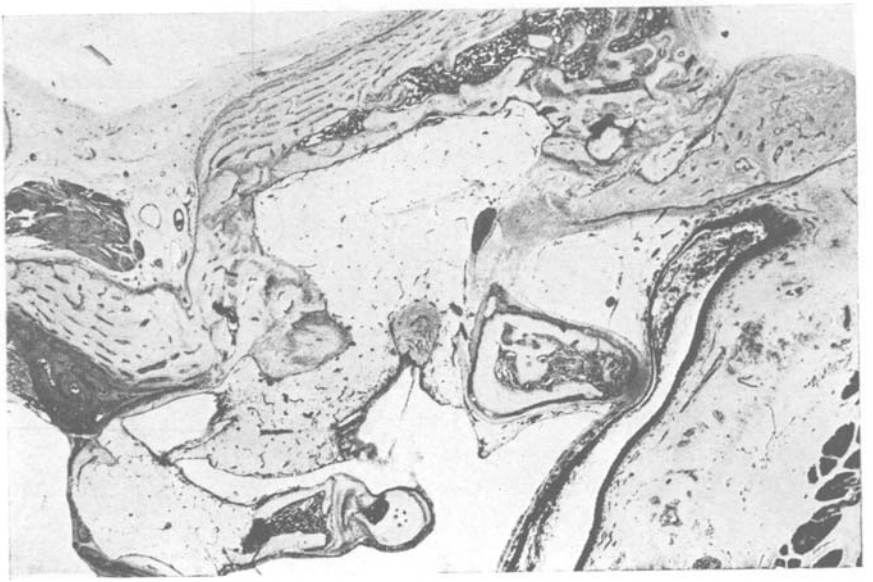

FIG. 4.
A vertical section through the middle ear of a new-born. Submucous connective tissue fills the incompletely pneumatized attic.

ingrowing prickle cells in the epidermis of Shrapnell's membrane which was neither indrawn nor perforated. He considered that this epithelial proliferation was caused by an inflammatory stimulus such as a prolonged attack of otitis media. In W. Lange's opinion these proliferating columns of basal cells grow into the submucous connective tissue of Prussak's space, thus forming the basis of the cholesteatomas. W. Albrecht and M. Schwarz, have pointed to the importance of connective tissue rests in the epitympanic space in the formation of Shrapnell's cholesteatomas. The development of the middle-ear cleft proceeds in two stages. As we could show, in a first stage bone cavities are formed; these cavities are pneumatized in a second stage. We have also been able to show in 1937 and in 1939 that in many new-born and very young infants extensive deposits

of submucous connective tissue fill the incompletely pneumatized attic and epitympanic cells (Fig. 4). Infants with this congenital "mucosal hyperplasia" are particularly liable to attacks of acute catarrhal inflammation of the middle ear. This recurrent inflammatory process may stimulate the active proliferation of basal cells in the pars flaccida of the drum as suggested by W. Lange. It may also facilitate the growth of columns of basal cells into the depths of the submucous connective tissue in the attic and thus initiate a cholesteatoma.

At the International Congress in Washington, D.C. (1957) I mentioned some observations which support W. Lange's view.

(1) The structure of the matrix of cholesteatoma with large drum defects and those with small upper marginal perforations are histologically identical with stratified squamous epithelium of the skin.

(2) The matrix of both types of cholesteatomas has the same tendency to active invasive growth, while the mucosa, submucosa and bone of the middle ear react to this growth in much the same way in either case.

(3) In the normal human fœtus of 7-9 months and in very young infants, the upper drum margin shows regularly a greatly augmented epidermis. The growth of the epidermis at the junction with the external auditory meatus amounts to a hyperkeratosis and acanthosis.

(4) As a result of low grade inflammation in the middle ear the growth of the meatal skin is activated. These findings imply that cholesteatomas with small upper marginal perforations as well as with large marginal defects of the tympanic membrane originate by immigration of squamous epithelium from the drum or the auditory meatus.

Differences in the early development, growth and the risks of complications in both types of cholesteatoma depend on the intensity of the inflammatory stimulus. In cases with large drum defects, arising in the course of a necrotizing otitis media in infancy, the skin of the auditory meatus receives a powerful stimulus. The squamous epithelium grows quickly through the large perforaton to invade the submucosa of the middle ear. This active growth is, however, confined to soft tissues and does not attack bone. The bone itself responds to the strong inflammatory stimulus by laying down protective bony lamellae and by a rapid ossificatior of the submucous connective tissue. The actively growing cholesteatomas are thus soon deprived of their mesenchymal basis. The reinforced sclerotic bony walls (Fig. 5) protect the internal ear and the endocranium at least as long as the dead cholesteatomatous material is extruded either spontaneously or therapeutically through the large tympanic perforation.

In Shrapnell's cholesteatomas, however, the initial growth of the basal cells from the still intact pars flaccida is probably much slower and intermittent. This corresponds to the much weaker stimulus of recurrent catarrhal middle-ear inflammations in early childhood. The slowly growing

FIG. 5.
Otitis media chronica cholesteatomatosa in the case of a 35 year old man. The cholesteatomatous matrix is lining a wall of sclerotic bone.

FIG. 6.
Shrapnell's cholesteatoma in the case of a not-operated 18 year old boy.

fingers of the early Shrapnell's cholesteatoma, nevertheless, extend deeply into the abundant connective tissue of the attic, while the drum remains intact. As the bony reaction is also mild in these cases the connective tissues become only slowly ossified. Long tubes of cholesteatoma grow out to the not yet pneumatized cell tissues before the appearance of a perforation. By the time a small, upper marginal perforation forms, the disease is too extensive to be managed conservatively. Having finally used up all the available submucous connective tissue (Fig. 6), the cholesteatoma fills the narrow spaces around the attic, becomes infected and causes a pressure absorption of the neighbouring bone. The infection cannot be reached through the small perforation and will continue to extend into the cranium. Thus cholesteatomas with small upper marginal perforations are more dangerous by virtue of the local conditions which facilitate the deep penetration of the matrix while diminishing the protective reaction of the surrounding bone.

One hundred and twenty-four temporal bones obtained from cases of cholesteatoma, acute otitis media and from normal young children were examined histologically by serial section. A search for evidence of embryonic cell rests or areas of metaplasia in the mucosa of the middle ear proved fruitless. While the occurrence of such cell rests or metaplasia is possible, it is at best very rare, while cholesteatomas are encountered commonly.

The suggestion was made by English workers that cholesteatomas may arise as a result of cholesterol granulomas. A. Tumarkin aptly observes that "the relation of cholesterol granuloma to cholesteatoma has given rise to much argument and the problem has been confused by a failure to define terms". The confusion in fact arises from the conflicting clinical terms applied to the histologically well-defined cholesterol granuloma. G. E. Shambaugh Jr., when considering the clinical picture of cholesterol granuloma in 1929 described it purely symptomatically as *blue drum*. H. V. Forster in 1947 spoke of the black mastoid on the basis of the macroscopic appearance at operation. In 1954 R. R. Simpson coined the term black cholesteatoma for cases of black cellular mastoid and suggested that all types of cholesteatoma may arise from a cholesterol granuloma. In 1956 J. F. Birrell in association with G. Young produced a terminological variation from black cholesteatoma to black cellular cholesteatosis. Seventy-seven of J. F. Birrell's 11 cases had a typical cholesterol granuloma according to the histological evidence and the three illustrations. In none of those 11 cases was there any evidence of cholesteatomatous matrix. R. R. Simpson's suggestion that cholesterol granuloma is a precursor to cholesteatoma is possibly based on the following definition of cholesteatoma of the middle ear by A. Eggston and D. Wolff: "The lesion as found in the ear may be defined as an amorphous mass of desquamated epithelium, *cholesterin crystals* and wax, surrounded by layers of more

recently grown stratified squamous epithelium, the whole forming an invasive cast of the cavity in which it lies." According to very accurate chemical experiments performed by A. Fazi of Pisa, B. Simonetta (1949) was able to show the term cholesteatoma to be a misleading one. "In two cases of large cholesteatomas the cholesteatomatous masses in their entirety were subjected to chemical analysis and this latter in fact revealed the presence of cholesterol, but in such minute traces as to be negligible." If cholesteatomas were to evolve from cholesterol granulomas then the metaplastic transition would have to include the dissolution and absorption of the abundant cholesterol crystals present in the granuloma. The ability of the human organism to destroy sterols appears, however, very doubtful. In the opinion of S. J. Thannhauser, one of the greatest authorities of the cellular lipoid metabolism, "destruction of the sterol skeleton in the intermediary metabolism cannot be accepted as a fact until the enzymes which should be involved in the process are discovered". R. R. Simpson's suggested transition from a cholesterol granuloma into a cholesteatoma thus seems less likely on these grounds.

There are at least two varieties of cholesterol granuloma, namely the genuine cholesterol granuloma and that occurring in association with a chronic suppurative otitis media.

G. E. Shambaugh first described the rare genuine cholesterol granuloma with the following clinical characteristics. A gradual onset of a conduction deafness without any signs of inflammation, a blue-grey discoloration of the intact tympanic membrane and radiological evidence of clouding of the more or less pneumatized mastoid. These changes are caused by an accumulation of brown-black slime and the formation of very vascular granulation tissue (Fig. 7) in the middle ear and its cellular extension. The granulation tissue contains numerous cholesterol crystals and giant cells. The bony walls of the middle ear are as a rule not affected.

Six cases of genuine cholesterol granuloma were seen and operated on at the Zürich clinic in the last six years. The above mentioned granulation tissue, rich in cholesterol, was found in every case and verified histologically. In none of our genuine cases was there any histological evidence of a cholesteatoma arising or even a mucosal metaplasia which might give rise to a cholesteatomatous matrix as a result of the presence of the granuloma. During the six-year follow-up period none of the cases showed any signs of a subsequent cholesteatoma formation. Any ætiological relationship between the rare genuine cholesterol granuloma and the common cholesteatoma is, therefore, considered most unlikely.

The second type of so-called associated cholesterol granuloma is much commoner. B. Simonetta (1932) has described a large cholesterol pseudocyst formed in a radical mastoid cavity, some time after complete healing of the wound. Such smaller cholesterol granulomas not infrequently occur in chronic middle-ear infections with or without cholesteatoma. They are

usually distal to the main lesion in cells which have become shut off during incomplete sclerosis of the mastoid. These cells, which no longer communicate with the middle ear, often contain sticky brownish sludge on a thickened and partially granulating mucosa. The granulation tissue is again rich in blood vessels, cholesterol crystals and giant cells. In cases where cholesteatoma and cholesterol granuloma co-exist, the division between the two pathological processes is sharp and striking, both structurally and in the distribution of cholesterol.

This is illustrated by the case of a 23 year old man with a cholesteatoma in the right middle ear (Fig. 8).

In spite of the proximity of the granuloma and cholesteatoma there is no evidence in these cases of epithelial metaplasia or of any metamorphosis of the granuloma into a cholesteatomatous matrix. We agree with B. Simonetta's statement "that in such a case the two manifestations, cholesteatomatous and cholesterinic, co-exist, but remain separate; if they fuse at all the fusion is purely secondary when a cholesteatoma in its progression has eroded the bony walls of periantral cells which contained a cholesterol granuloma".

Thus biochemical, histological and clinical evidence all point against the possibility of cholesteatomas arising from cholesterol granulomas. As the ætiology of the latter still remains to be elucidated we would suggest that the histologically descriptive term of *cholesterol granuloma* of the middle ear be used rather than "black cholesteatoma" or "black cellular cholesteatosis".

In spite of the evidence presented in favour of the immigation theory enthusiastic supporters of the metaplasia school could object that all the histological material showed only proliferation of the basal cells in Shrapnell's membrane as a result of inflammation. Histological proof of the origin and development of cholesteatoma behind the intact drum is still lacking in man. Such evidence may be available in animal experiments.

Typical cholesteatomas in animals have been produced by the application to the external auditory meatus of cold or hot tar, benzpyrene, croton oil or olive oil or by injection of these substances into the middle ear (J. Berberich (1927), H. Hoshiya (1935), T. Ohta (1940), R. Schröer (1957)). The skin of the deep meatus proliferates in these experiments and grows into the middle ear through a myringotomy opening or spontaneous perforation. No signs of metaplasia of the mucosa of the middle ear were found on any occasion. In this connection a special mention must be made of the brilliant experiments performed by J. Friedmann, who infected the middle ear in guinea-pigs with *Pseudomonas pyocyanea* and with *Streptococcus pneumonicus*. He then found that "the stratified squamous epithelium from the external auditory meatus or from the tympanic membrane immigrated through the perforated tympanic

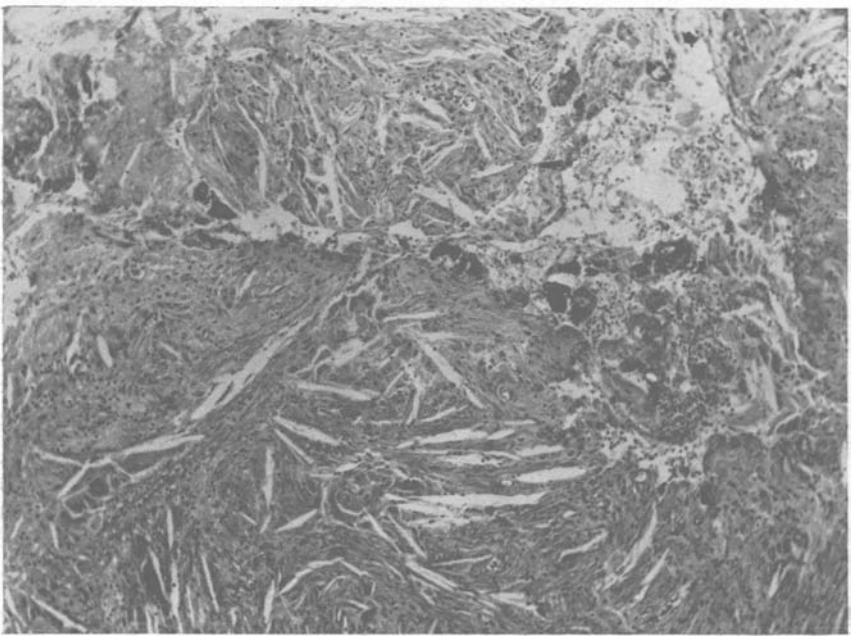

FIG. 7.
Genuine cholesterol-granuloma in the case of a 21 year old girl.

FIG. 8.
Otitis media chronica cholesteatomatosa in the case of a 23 year old man. Associated cholesterol-granuloma in the neighbourhood of the cholesteatomatous matrix.

membrane into the infected bulla of the guinea-pig, reproducing here a typical aural cholesteatoma". J. Friedmann's experimental findings fit in very well with J. Habermann's classical observations in necrotizing otitis media of infants. Review of the literature shows that a tympanic perforation was present in all the experimental cases of cholesteatoma. The cholesteatoma developing behind an intact tympanic membrane, which is of special interest to us, has not yet been reproduced experimentally.

In order to test this possibility, we have opened the aural bulla in guinea-pigs and stuck a mixture of talc and fibrin in several places onto the internal surface of the intact tympanic membrane. This caused a mild foreign body reaction and in several animals granulation tissue developed between the drum and the internal wall of the middle ear (Fig. 9). After 15-20 days there occurs in some animals an active ingrowing of the epidermis from the intact tympanic membrane into the newly formed granulation tissue. The invading columns of basal cells divide into branches (Fig. 10) and the desquamated stratified squamous epithelium forms typical cholesteatomatous masses. Thus a mild inflammatory reaction resulting from chronic irritation may produce a cholesteatoma behind an intact tympanic membrane in the guinea-pig. Further details will be given in a special paper. There is no doubt that this experimental cholesteatoma results from an immigration of the squamous epithelium of the tympanic membrane stimulated by an inflammatory reaction. This makes the proposition that human aural cholesteatomas behave in much the same way all the more likely. The initial and the final stages of immigration of stratified squamous epithelium have already been determined in man. However, the intermediate stage of cholesteatoma formation behind the still intact tympanic membrane, which we have been able to demonstrate in experimental animals, has not yet received histological confirmation in man. In common with J. Friedmann we have not been able to find any evidence of mucosal metaplasia or cholesterol-granuloma in any of our guinea-pigs.

Clinical and histological study of normal and pathological human ears, supported by animal experiments, leads to the conclusion that as a rule all types of cholesteatoma of the middle ear develop by immigration of stratified squamous epithelium from the epidermis of the external auditory meatus or the tympanic membrane. Within the middle-ear cavities the active growth of the matrix is enhanced by submucous connective tissue filling the incompletely pneumatized attic and epitympanic cells. As seen in the guinea-pig, the cholesteatomatous matrix may also continue to grow within newly formed granulation tissue.

The practical application of the immigation theory to the elimination of the risks of cholesteatomas, achieving a dry ear and improving the hearing must now be considered.

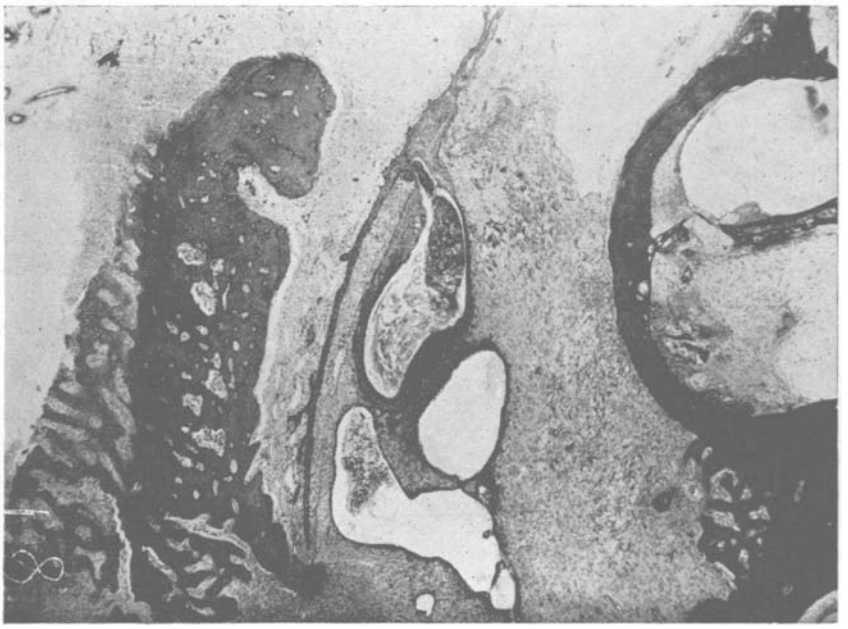

FIG. 9.
Vertical section through the middle ear of a guinea-pig. The external meatus is narrowed to a slit but the tympanic membrane, is intact. Active ingrowing of the epidermis from the intact tympanic membrane into the newly formed granulation tissue.

FIG. 10.
Higher magnification of Fig. 9 (vertical section through the middle ear of a guinea-pig). Experimental cholesteatoma develops behind the intact tympanic membrane.

Cholesteatosis of the Attic

The dangers of cholesteatoma depend on the growth of the matrix into the middle ear and its further multiplication there. In order to eliminate this dangerous process it is essential thoroughly to eradicate the matrix, the submucosal connective tissue which facilitates its spread, and all the granulation tissue in the surrounding cells. Complete removal of the cholesteatoma can only be achieved surgically. As cholesteatomas usually begin in childhood, the sooner they are removed the less extensive will be the resultant damage to the ear. The most important therapeutic principle is, therefore, *all forms of cholesteatoma should be treated surgically as early as possible.*

The cholesteatomatous matrix grows in depth, affecting all the cell groups still available in the mastoid process of the child. In order to eradicate the disease all the cells must be meticulously opened and cleared out. This important step in the operation is often technically difficult, especially in the sub- and retrofacial and the hypotympanic peritubal cells. We attempt to achieve the last two aims of treatment, namely the establishing of a dry ear and improving the hearing by means of tympanoplasty. In earlier years to ensure a dry ear, many methods of shutting off the Eustachian tube through plombage of its tympanic opening were tried, often without success. Today the newly fashioned cavum tympani is shut off more easily by means of a new tympanic membrane. This new membrane fashioned from a skin flap of the anterior meatal wall bridges the hypotympanum and is inserted along the external semicircular canal. The membrane covers the facial ridge, and passing forwards and upwards it will lie somewhat lateral to the original tympanic annulus on the anterior meatal wall.

Improvement in hearing depends on the patency of the tube, integrity of the ossicular chain and the function of both the inner-ear windows. A stenosed Eustachian tube is dilated with silver sounds and it is held open by passing a small acryllic tube down and out through the nose. In order to keep the newly fashioned cavum tympani in communication with the round window, it is inflated with air through the indwelling acryllic tube for the first ten days. Unfortunately the ossicular chain is very often interrupted by the cholesteatoma, especially at the point where the long process of the incus articulates with the stapes. Thus only the stapes or sometimes only the mobile footplate remain available for the conduction of sound. Accordingly the new tympanic membrane flap is either placed directly onto the stapes or connected to the footplate by the interposition of a columella. Finally the niche of the round window, which is frequently overgrown, must be freed if an improvement in the hearing is to be achieved. Further technical details of the tympanoplasty are then dealt with as they arise in the course of the operation.

Fig. 11 gives the surgical results which have been achieved in 88 cases of aural cholesteatoma.

Number of cases		88 = 100	per cent.
(1) Endocranial danger definitely eliminated		88 = 100	per cent.
(2) Ear dry		78 = 88·6	per cent.
(3) Tympanoplasty successful		69 = 78·4	per cent.
(4) Hearing improved		44 = 50·0	per cent.
(*a*) More than 20 db.	10 = 11·4	per cent.	
(*b*) 10-20 db.	19 = 21·6	per cent.	
(*c*) Until 10 db.	15 = 17·0	per cent.	
(5) Hearing unchanged		25 = 28·4	per cent.
(6) Hearing worse		19 = 21·6	per cent.
(*a*) Until 10 db.	11 = 12·5	per cent.	
(*b*) More than 10 db.	8 = 9·1	per cent.	

FIG. 11.

Better ear surgeons will get better results.

Summary

Cholesteatomas in the middle ear are classified in a larger, less dangerous group with large upper marginal perforations and in a smaller more dangerous group with small upper anterior or posterior perforations.

Clinical and histological studies have shown that as a rule cholesteatomas with large and with small upper marginal perforations develop by immigration of stratified squamous epithelium from the external auditory meatus or tympanic membrane. This process takes place in some cases during acute necrotizing otitis media through a large upper marginal tympanic perforation. Aural cholesteatomas with small upper marginal perforations develop insidiously as a result of recurrent acute catarrhal inflammations of the middle ear in childhood. The basal cells of the intact pars flaccida of the ear drum multiply and grow into the submucous connective tissue filling the incompletely pneumatized attic, or into newly formed granulation tissue, where they continue to grow into a cholesteatoma. These cholesteatomas, which have primarily originated behind an intact tympanic membrane, usually break through into the external auditory meatus and give rise to a small, upper marginal perforation. The formation of cholesteatomas behind an intact tympanic membrane has been reproduced experimentally in guinea-pigs by setting up a mild inflammatory reaction in the middle ear.

The risk of intracranial complications is ever present in all types of cholesteatoma because of the invasive tendency of the matrix. In order to eliminate this risk and to arrest the persistent otorrhoea, early surgery is indicated. Tympanoplasty offers the best means of improving or at least preserving the hearing, depending on the integrity of the ossicular chain, the state of the round window, and the patency of the Eustachian tube.

The operative results obtained at the Zürich E.N.T. Clinic during the last two years are presented.

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